Characterization of Structural and Mechanical Properties of IPSC-derived Cardiomyocytes for Future Application to Familial Cardiomyopathies

Paige Cloonan
Washington University in St. Louis

Follow this and additional works at: https://openscholarship.wustl.edu/wuurd_vol12

Recommended Citation
https://openscholarship.wustl.edu/wuurd_vol12/36

This Abstracts A-I is brought to you for free and open access by the Washington University Undergraduate Research Digest at Washington University Open Scholarship. It has been accepted for inclusion in Volume 12 by an authorized administrator of Washington University Open Scholarship. For more information, please contact digital@wumail.wustl.edu.
Familial cardiomyopathies are a leading cause of sudden death in young people, and as of now, there is no known cure. While mouse models and patient heart tissue have been extraordinarily useful in the study of the disease pathogenesis, the physiological differences between mouse and human cardiomyocytes can introduce many inconsistencies in disease presentation. Additionally, tissue from patients with the disease is difficult to obtain and it has undergone many compensations that make it difficult to understand the early stages of the disease pathogenesis. To develop a new model of the disease, we are using human stem cell derived cardiomyocytes bearing mutations that cause familial hypertrophic cardiomyopathies. We have introduced these mutations using CRISPR. Wild type stem cells were differentiated into cardiomyocytes. Immunostaining helped assess sarcomeric organization and traction force microscopy provided a tool to measure the force produced by single cardiomyocytes. We are now beginning to apply the same tools to study a hypertrophic cardiomyopathy disease causing mutation in troponin T, R92Q.